KANSAS MEDICAL JOURNAL.

VOL. VII. TOPEKA, KANSAS, SATURDAY, NOVEMBER 30, 1895. No. 48.

ORIGINAL ARTICLE.

Pus Formation—Its Field and Factors.

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Read before the Kansas Medical Society, at Topeka, May 16. 1895.

The formation of pus being one of the most frequent and familiar of pathological phenomena, its presence until comparatively lately had not caused the majority of practitioners to reflect fully upon the factors to its production. It has been, it would seem, something like the idea of the Irishman, who, when the grandeur of Niagara and its wonderful falls were pointed out to him and his mind directed to the immense body of water pouring over the rocky edge, asked, "What is to prevint it?" Do the majority of practitioners, even now, do all they should to prevent the Niagara of pus, or fully appreciate the pathological meaning of its presence? I believe they do not.

Moreover, I am convinced that many practitioners, and I include a large number of general and special medical men of ability, are more or less free from all acquaintance with many of the facts and theories which modern pathology furnish in regard to pus formation and to other pathological advances. And I am not only convinced that this is true from conversing with physicians, but also as a derivation from my experience in discussions with them in medical societies. I would not include, as a rule, the younger men in this rating, for most of these have been trained in later views; but I refer more especially to those who have been longer in the profession, many of whom have become so "set" in their views in pathology, that when anything new is offered to them are willing to throw it over without investigation as to its probability, and without even going over the facts presented to them so as to familiarize themselves with points claimed. In other words, a rejection is made from ignorance.

Holding this view, I believe that papers on modern pathological facts ought not to come amiss, hence my remarks to-night.

The object of this paper is to consider pus formation, its field of work and its factors. And it may be well here to state that we shall not include in our consideration the fluid of chronic or cold abscess which is not a collection of pus and should not be so called, but in reality is an emulsion, its factors being loose fat, few cells fattily degenerated and broken down and emulsified with an effused albuminous fluid, plus disintegrated tissue.

The broad statement can now be made that pus can only be formed in or on living tissues by microbic action. I am aware of course that it is possible by the injection into the tissues of certain irritants and caustics and strong acids to produce a limited amount of innocuous pus, but that this fluid will not produce pus if innoculated intoo ther individuals; and, moreover, will not extend its own immediate limits or quantity in the same individual; nor will it produce any constitutional symptoms in the individual in which it is produced.

A typical picture of pus formation is found in cases of ulcerative endocarditis, for in the muscular wall of the heart in these cases may be found minute abscesses quite near, generally, the endocardial surface.

On a section of a portion of a heart wall containing such an abscess we can observe a small gray or yellow point, with a zone

of reddened (congested) tissue just around it. If one of the portions containing an abscess is hardened and stained, we shall find the following conditions presented to us:

By the aid of a low power objective, we can notice in the center of the purulent focus one or more minute blood vessels with the lumen more or less filled by a thrombus, which latter will have a microbic accompaniment and many leucocytes. The vessel wall will be indistinct and more or less structureless. The lymph space around the vessel, and a zone of tissue immediately beyond, will also show this structureless picture not normal to it. In these zones we may or may not find microbes, but beyond them we find an enormous number of leucocytes, infiltrating the muscular tissue everywhere. These leucocytes are rapidly dying and becoming pus corpuscles. In addition to the leucocytes we find a few larger cells—proliferated and unproliferated connective tissue cells—whose source and function we shall refer to later. Beyond the periphery of the abscess, but close to the latter, it will be found and sharply defined, too, that the tissues are in normal condition save this, that leucocytes are infiltrating even the normal tissues there for a certain distance, this normal tissue contrasting, however, markedly with the broken down and disintegrating muscular tissue of the abscess center proper. (Woodhead.)

With a high-power objective we can obtain with each field observed more complete details of the changes. We can see the cocci in large numbers in the thrombotic vessel. Beyond the vessel some few details of structure can be seen. The staining, however, is imperfect, but we can observe the disintegrating muscular fibres and can make out some of the leucocytes, and also a number of nuclei, which latter are from disintegration of other leucocytes. In the zone beyond this, which may be called one of coagulation necrosis, we find the leucocytes, many of which are more or less active, as shown by their taking the stain, and occasionally one still having amœboid movement.

At the periphery of the abscess we find the larger connective tissue cells, which are to be differentiated from leucocytes; these are by some pathologists called fibroblasts. We can also see in the body of the abscess more readily with this power the disintegrated muscular tissue. And beyond the margin of the abscess leucocytes infiltrating the otherwise normal muscular interspaces.

Allowing for the difference, anatomically, in the attacking point—that is whether the microbes work first from within or without the abscess. We may observe practically a nearly similar condition where pus is formed on a mucous or a serous membrane. Take the latter first, and for this the omentum furnishes a good field. Recalling the histological arrangement here, we find the endothelial covering with the stomata and connective fibres and connective tissue cells, and last the blood vessels. With this ground understood we can see here, by microscopic aid, the leucocytes exuded from the engorged blood vessels and also the microbes. We shall observe the endothelial cells proliferating and separating, and we shall find the connective tissue cells in the lymph spaces also multiplying and separating. The leucocytes and proliferated endothelial cells are exuded in and upon the serous membrane and become pus cells by microbic action.

We may in gonorrhœal ophthalmia also see the destructive and pus producing effect of the gonococci. The microbes here produce exfoliation of the epithelium and can be seen in the epithelial cell and intercellular substance and may be traced as far beneath the epithelial covering as the submucosa, from the vessels of which latter, leucocytes are exuded or escape to become included in the protoplasm of the gonococci, and later to die and become pus cells.

In several of the other mucous membranes the gonococci produce a similar result, but the work of the gonococci there also, as well as in the conjunctiva is confined to a comparatively superficial field; the deeper pus collections occasionally found in gonorrhœa and as a sequela to it both in the male and female must largely, if not wholly, be dependent on a microbic coworker—of which the gonococcus is the essential pioneer. I shall allude to this again, however, later.

We shall find the same result as far as the phenomena attending pus formation is concerned in the non vascular tissues—or rather, to state it more definitely, in tissues free from blood vessels. Take the cornea for instance. Here a centrally located abscess may be produced by innoculating with a pus producing microbe like the staphyloccus p. a., and in this instance the leucocytes which form the pus cells are derived from the lymph channels, both the wandering and the fixed cells furnishing their quota to the central assembly. Later the circumcorneal blood vessels throw into the work leucocytes which pass through the corneal lymph spaces to join the throng directly—some of them perhaps lingering on the way to cloud the cornea in the abcess' vicinity. In the vitreous humor the same pathological changes occur—the leucocytes traveling to the involved focus in this case, possibly still farther from their distant habitat in lymph space and blood vessel, with a chemiotoxic power that is even more remarkable than in some of the other tissues similarly involved.

We can observe the same changes which produce pus in other parts or organs of the body; in the joints, in large organs like the liver and elsewhere in pyemic conditions, the actual pus formation is practically similar.

Having briefly described an abscess and its surroundings, and also other pus collections with their environment, we may now consider more in detail the conditions which lead up to pus formation in general. This we can the more readily, and perhaps the more satisfactorily, do by referring to the abscess or localized pus formation again first. We observe here the thrombotic vessel with the microbes, and notice that the vessel wall and a zone beyond it yields a structureless picture to the microscope. The tissues of the zone have been killed—probably digested by the toxines of the microbes. In this zone we find leucocytes and a few larger cells and debris. The leucocytes here scarcely take the stains, but we may find in those which do that then the protoplasm contains their bacteric enemies. Now while it is difficult, if not impossible, to say how the microbes affect the death of the tissues and the leucocytes, there can be no doubt of their doing so. The theory that in their metabolism they excrete a toxine which kills and afterwards digests the tissues first and the leucocytes later, would seem to be the most satisfactory. At any rate, we actually find in pus, as we shall see later, peptones and other derivatives of digestive action. (Woodhead.)

In the zone beyond that which is structureless the microbecidic work is more confined to the leucocytes and the fibroblasts, perhaps; still, even here, if the microbes be very numerous, their destructive work on tissues still goes on. We find the leucocytes have come to the site of the portion involved in large numbers and their function in neutralizing the toxine, killing the bacteria and digesting and removing them (probably they have the power of all these added to that of the fibroblasts), in most instances is unsuccessful in bringing about a cessation of the purulent formation. At any rate the leucocytes and the proliferated connective tissue cells form a zone around the microbes, and if numerous enough, stop microbic multiplication and action and bring pus formation to a halt.

We may now consider two of the theories as to how this result obtains:

First, that of Metschnikoff, and known as phagocytosis. The author of it has shown much industry and intelligence in arriving at his conclusion. He divides the phagocytes into "two broad groups of *fixed* phagocytes (endothelial cells, etc.,) and free leucocytes." And he calls attention to the fact that all leucocytes are not phagocytes; but of that later. The theory in a nutshell is "that in the property of its amœboid cells to include and destroy micro-organisms, the animal body possesses a formidable means of resistance and defence against these infectious agents." Now, ingenious as the theory is, there can be offered several objections to it. It may be partly true, but it certainly is not completely so. One of the facts which Metschnikoff brings forward among others to support his theory, viz.,

that the phagocytes take the microbes within their protoplasm is true, and it is mainly upon this one fact that he seeks to establish his whole theory for the destruction of microbes. There can be no doubt that phagocytosis occurs, that is admitted and this process may have its part, possibly even a large part in causing destruction of the microbe. But it would seem that the leucocytes and perhaps their coworkers, the endothelial cells, first have the power of either killing or very much weakening the micro-organisms *before* they take the latter into their protoplasms. In other words that a large part of the phagocytic function is that of scavengers. And we mention here in support of this one or two clinical facts. It is known in relapsing fever, that during the pyrexia the spirilum which produces this fever exists in the blood serum only and not in the blood corpuscles, while in the apyretic stage the microbe is only found in the corpuscles and not in the serum, indicating that while the micro-organism is in its most active state the phagocytic power is not exhibited and *vice versa.* I know that Metschnikoff offers as against all opposers of this view that living microbes in certain instances are more readily taken up by phagocytes when injected into an animal than are the same microbes which have been killed before injection. But of this objection it may be urged that many of the laboratory microbes have as part at any rate this necessity for their destructive work, namely, that they must first become stationary; and also to allow of any action by leucocytes they must be brought to a standstill; and this the living microbe is more apt to do by making a thrombotic dam to the blood current in one or more of the blood vessels; and that when stationary, the leucocytes can the more readily swarm round them and destroy them. While the dead microbes are more apt to circulate indefinitely and without any concentrated leucocytic action upon them. Moreover, Sternberg states that microbes which can be found in phagocytes, are rarely so found in the early stages of the disease which they produce.

Another theory, and probably the most satisfactory of all which have been offered, is based upon that of Behring in regard to immunity. This involves the view "that some substance is formed in the body of the immune animal which neutralizes the toxic products of the pathogenic micro-organism." (Sternberg.)

Now this theory is, of course, mainly applicable to general or systemic effects of microbes, and it would seem to be the underlying fact which is applicable not only in so far as the destruction of the toxines are concerned, but should also include a power destructive of the microbes which produce the toxines. Now reasoning from the general to the particular, we may apply this theory for the destruction of the pus microbes. And in seeking as to where the antitoxic power resides, we need hardly look further than the leucocytes, and perhaps also the connective tissue cells, the fibroblasts. As I have previously stated, no one doubts the fact that these two morphological elements are capable of taking microbes within their protoplasms; *but it would seem that they can only do so after the micro-organisms are either disabled and dying or dead.* And it would also seem that in all probability the leucocytes at least are capable of throwing out a "digestive fluid or some material detrimental to the existence of micro-organisms may prepare the organisms, killing and even partially digesting them, taking them into their protoplasm as inert matter and there continuing the process of digestion." (Woodhead.)

This theory appears to be more tenable than that of Metschnikoff's, certainly more complete.

It can be readily appreciated of course, in their work of saving the tissues from the attack of the microbes, that the leucocytes succumb in large numbers and that they as well as the surrounding tissues perhaps have before them, but given that the individual microbic working is local, that there is no general toxic effect, and that the leucocytes are sufficiently numerous, the pus micro-organisms sooner or later are not only barred from invading new zones, but are

also destroyed in and removed from zones they have occupied by the leucocytes, the killed and injured leucocytes furnishing, in the main, the morphological elements of the pus.

Bearing on the germicidal properties of leucocytes, Vaughan, in his remarkable paper on nuclein in the *Medical News*, December 23, 1893, states that "Blood serum contains a nuclein. . . . The origin of the nuclein found now for the first time in blood serum is an interesting question. Does it come from the disintegration of the polynuclear cells, or shall we regard certain white corpuscles as unicellular organs whose function it is to secrete this nuclein?"

On these two theories, I will quote from Sternberg: "Numerous experiments have been made within the past two or three years with a view of determining whether pathogenic bacteria are, in fact, destroyed within the leucocytes after being picked up, and different experimenters have arrived at different conclusions. In the case of mouse septicemia already alluded to, and in gonorrhœa, one would be disposed to decide from the appearance and arrangement of the pathogenic bacteria in the leucocytes, that they are not destroyed, but that, on the other hand, multiply in the interior of those cells, which in the end succumb to this parasitic invasion. In both of these diseases we find leucocytes so completely filled with the pathogenic micro-organisms that it is difficult to believe that they have all been picked up by a voracious phagocyte, which has stuffed itself to repletion, while numerous other leucocytes from same source and in the same microscopic field of view have failed to capture a single bacillus or micrococcus. Moreover, the staining of the parasitic invaders, and the characteristic arrangement of the gonococcus in stained preparations of gonorrhœal pus indicate that their vitality has not been destroyed in the interior of the leucocyte or pus cells, and we can scarcely doubt that the large number found in certain cells is due to multiplication in situ rather than to an unusual activity of these particular cells. But in certain infectious diseases, and especially in anthrax the bacilli included within the leucocytes often give evidence of degenerative changes, which would support the view that they are destroyed by the leucocytes, unless these changes occurred before they were picked up by the leucocytes, as is maintained by Nuttall and others. We cannot consider this question definitely settled."

I will quote from Metschnikoff, which, according to my view, militates against rather than confirms this bacteriologist's theory that the phagocytosis is *the* factor in recovery and immunity:

"Where a given species of animal is especially sensitive to the onslaught of one or other micro-organism, there during the course of the disease the phagocytes are inoperative, including none or almost none of the bacteria. On the other hand, when by previous vaccination these animals have been rendered refractory, their phagocytes have acquired the property of including the same bacteria." (Sternberg, p. 21.)

This clearly indicates that while the bacteria are most virulent the leucocytes have first to injure or destroy them before admitting them into their protoplasm. In other words, that phagocytosis is in many instances a secondary step in bringing recovery.

The condition of acquired immunity from or of cure of infectious diseases cannot in all cases depend on phagocytosis, for we have in some of these infectious diseases the fact that the microbes, say, for instance, in diphtheria, are not found in the circulation, but are local. Their toxines of course circulate, and it is in that disease injections of serum destroy the toxines, probably, first, and prevent the multiplication of their microbic causes later. Moreover, the symptoms of the disease, practically the disease itself, is produced by injections of filtered cultures. What part then can phagocytosis play there?

Further, "Ogasata and Iasuhara in a series of experiments made at the Hygienic Institute at Tokio, (1890), discovered the important fact that the blood of an animal immune against anthrax contains some substance which neutralizes the toxic product

of the anthrax bacillus. When cultures are made in the blood of dogs, frogs or white rats, which animals have an immunity against anthrax, they were found not to kill mice innoculated with them. Further experiments showed that mice innoculated with virulent anthrax cultures did not succumb to anthrax septicemia if they received at the same time a subcutaneous injection of a small quantity of the blood of an immune animal. So small a dose as one drop of frog's blood or one half a drop of dog's blood proved to be sufficient to protect a mouse from the fatal effect of an anthrax innoculation." (Sternberg, p. 256.)

"Behring and Kitaoata have obtained similar results in their experiments upon tetanus and diphtheria." (Sternberg.)

In considering the field of pus formation, it may be said that probably no tissue can be excepted; certainly no structures which include connective tissues can be. Of course some tissues offer a more ready receptacle for pus formation than others; and this is especially true of the looser so-called cellular tissues.

There is one important organ of the body that is scarcely ever invaded by pus and that is the spleen. I have every reason to doubt its frequent formation here unless the microbic cause is reinforced by trauma. In all of the post mortem examinations of bodies dead from pyemia which I made during the war it was observed, while pus could be found in all of the joints, in all of the important organs and viscera, I cannot recall a single case in which pus was discovered in the spleen. I met with one case of splenic abscess after the war which had a traumatic cause as one of its factors

As we shall see later, certain of the pus-producing microbes can only grow in or on a certain tissue; and what is also true is the fact that certain pus producing micro-organisms when put into one tissue will produce simply an abscess, and in another structure will cause death. Cheyne has proved, for instance, that the proteus vulgaris, if injected into the subcutaneous tissue of a rabbit's back, will cause an abscess; while if it is introduced into the dorsal muscles will cause death. Acute infective osteomyelitis seems to be limited to growing bones. The peritoneum, as every one is aware, can be a possessor of pus, yet considering the fact in these days of numerous cœliotomies, how rare is pus, and while every modern laparotomist tries his best to make his intraperitoneal operation as aseptic as possible, this is not always, many times cannot be, completely possible; still the operator knows that he has within the peritoneum the aid of a great absorbent sac, which if not too many microbes be permitted to remain in it, that this great lymph space will most hospitably absorb and safely provide for a large number of some of the pus cocci without damage, showing after all that pus microbes and pus here would not be as readily multiplied as if placed perhaps in ordinary connective tissue.

It will be essential in referring to the pus factors to recall some few histological points; and first as to the leucocytes in the blood, "we find:

1. Small lymphocytes, which are smaller than the red corpuscles. They have a readily stained large spherical nucleus which nearly fills the cell.

2. Large lymphocytes which some histologists consider as an advanced stage of the above. They have a large nucleus surrounded by a well defined protoplasmic zone. These two forms make up 25 per cent. of the leucocytes in blood.

3. Mononuclear elements or transition forms—one nucleus, not spherical, but with a depressed center.

4. Polynuclear leucocytes smaller than the last; nucleus with several lobes or several nuclei. They make up 70 per cent. of all the leucocytes in the blood.

5. Eosinophilous cells—The nucleus stains less deeply than the last. The granules of the protoplasm stain very deeply with eosin. This cell is but sparsely found in healthy blood." (Sterling.)

Now, as also furnishing morphologous elements to the pus we must perhaps add the plasma cell of the connective tissue, and we find in the connective tissue also the granule cell, which latter is quite an active

element in pus formation. This is the eosinophilous cell and is more frequent in the connective tissue than in normal blood.

The micro-organisms which produce pus in man are not of many species, and Sternberg states: "Of these the two most important, by reason of their frequent occurrence and pathogenic power, are the staphylococcus pyogenes aureus and streptococcus pyogenes; next to these come staph. p. albus, and the following species are occasionally found: Staph. p. citreus, staph. cereus flavus, staph. c. albus, micrococcus tenuis, bacillus pyogenes fœtidus, micrococcus tetragenus, micrococcus pneumoniæ crouposæ. Two or more species are often found in the same abscess; thus Passet in 33 cases of acute abscess found staph. aureus and albus associated in 11, albus alone in 4, albus and citreus in 2, streptococcus p. alone in 8, albus and streptococcus in 1, and albus, citreus and streptococcus in one. . . . Robb and Grisky have shown that under the most rigid antiseptic treatment migro-organisms are constantly found attached to sutures when these are removed from wounds made by the surgeon, and that a skin abscess frequently results from the presence of the most common of these organisms—staph. epiderm. albus." (Sternberg.)

Of these microbes the micrococcus pneumoniæ may be eliminated probably, and while it is frequently found in pus from middle ear and other sites, it is probably accidental and plays but a small part in pus making.

We must add that the most active and malignant pus producer is the gonococcus. This microbe has, from the difficulty of definite and absolute demonstration, been the cause of much discussion. Still from the fact that it is found in the pus cell, that it does not stain by the Gram method, we should have no difficulty in recognizing it. "No similar picture is presented by pus from any other source, with the exception of that from a form of puerperal cystitis, which Bunn has described. But in this the diplococci contained in the pus cells were to be distinguished by the fact that they retained their color when treated by Gram's method. Owing to the difficulty of cultivating this micrococcus, and the importance under certain circumstances of not making a mistake in its diagnosis, these characteristics are of exceptional value." (Sternberg.)

There are a few conditions which should be remembered in reference to all the above-mentioned microbes. The ordinary pus producers can only manifest their pus producing power after gaining admission beneath the surface. And if they gain entrance to a blood vessel so long as they circulate with the blood no pus is formed; but if there be an injured or diseased focus they may cause thrombosis there and set up their pus factory in the surrounding tissue. On the surface of the cutaneous epidermis or of the mucous epithelia an abrasion must occur for them before their work can begin; were it otherwise there could be no freedom for man from surface pus formations, it being well known that one of these species of microbes, the staph. epiderm. albus, is a normal inhabitant of the cutaneous surface at all times. On the contrary the gonococcus needs only a contact with certain mucous membranes to begin its despicable work. And while this microbe of Neisser may have and undoubtedly often does have assistants in destructive work, such as the staphylococci, still this latter species cannot begin pus formation on a mucous membrane until the gonococcus has previously prepared the mucus surface by abrading it. Were it otherwise the ocular and other mucous membranes for the most part would never be free from pus. But in all probability the part played by the staphylo. when found with the gonococci is a secondary one in most instances, at any rate in the conjunctiva. In the mucous membranes of the genitalia with the gonococcus for a pioneer, a very much deeper-seated work of destruction may go on in the formation of abscesses and other pus accumulations, with the staphylococci as a follower or coworker.

Both streptococci and staphylococci are found in pus from the middle ear, but here the preceding inflammation has abraded the mucous membrane and given these microbes access for deeper work.

As to how the microbes destroy the leu-

cocytes and other cells which come to the front to battle against them can only be explained by the micro-organisms forming "either of a poisonous chemical substance which acts as an irritant, or by its direct digestive action on the protoplasm brings about a process of degeneration which has been compared to that produced by the enzymes when acting on albuminoid materials; in the immediate neighborhood of these organisms both highly organized cells and connective tissues are in fact digested." (Woodhead.)

Frequently there is some diminished resistance, the result of trauma or other depressing causes which precipitates the pus microbe at the point. We have this for instance in ulcerative endocarditis, with its precedent abscess of the heart wall. Ribert's experiments would seem to prove this. He injected cultures which produce the lesion without effect until injury of the valves of the heart, then abscesses followed in the heart walls, such as I have described in the early part of thi paper. In a general way we can say that the extent of a pus formation may be taken as a measure expressive of the resistance of the part involved plus the virulence of the microbe. It might be formulated thus: The extent and severity of the pus collection is inversely as the resistance, directly as the virulence of the microbe.

As is generally known, pathogenic attenuation of micro-organisms or the reverse can be experimentally brought about, so it can be readily inferred that the increase or diminution of virulence of microbes in a given case may occur, in which there is also a lessened or increased resistance of the individual. Probably all the pus producing microbes are applicable to the above, which may be regarded as a law.

I believe that the fact of the attenuation of virulence and the ability to increase again the pathogenic force of a microbe has a very important clinical bearing. Take the gonococcus, and every careful genito-urinary surgeon recognizes that the majority of cases of gonorrhea treated leave him before a complete cure. The microbes in such cases are still to be found in the urethral follicles, attenuated in power, it may be, but capable of producing a sharp gonorrheal attack in the female. The male believes he is cured, marries, and carries in all probability the sentence of permanent suffering to his wife.

One other point as to the pus microbes, and an important one, too, is often shown and known to take place after other pathogenic but non-pus-producing micro-organisms have caused lesions. This is true in diphtheria, where we may have and do have pus produced upon the abraded pharyngeal mucous membrane, with deep purulent foci resulting, and with occasionally septicemia or even pyemia. This condition as a sequela of diphtheria might result in making nil the otherwise successful work of antitoxines. This fact has resulted in an effort to combine an antitoxine for pus microbes with the diphtheria antoxine.

In some cases of purulent peritonitis, too, we may have the colon bacillus as a forerunner, perhaps, of the other pus microbes. Possibly, also, of some the ordinary pus producing micro-organisms reinforce each other; at any rate more than one species is often found in the same pus from various sources.

Now, a few words as to the makeup of pus: This fluid, if taken from acute abscess, has very little odor, a specific gravity of about 1030, and is generally alkaline in reaction. "It contains from 10 to 15 per cent. of solid matter of which two-thirds are albumen." (Green.) In it may also be found peptones, leucin, etc.

If we examine recently formed pus microscopically we can observe that the pus corpuscles are in various conditions of degeneration. Some of them if we place them in the warm stage will perhaps still show the amœboid movement. Others are as quiet as death, though not too far gone in degeneration to prevent them taking the stains. Others while still preserving shape show signs of a fatty break up and osmic acid will show the fat. We may also see some granular debris.

The microbes causing the pus we examine may be found if the fluid is very recent, but

generally the microbes are too far gone to take the staining in large numbers. This does not apply to gonorrheal pus for obvious reasons. Beyond a doubt the majority of the pus corpuscles are leucocytes. We find a few of the large connective tissue corpuscles, but they are as a rule degenerated. We find also shreds of broken down tissues.

How Does Antitoxine Act?

The modus operandi of diphtheria antitoxine is yet unexplained. If it contributes toward cure it does so by counteracting the poison or by facilitating nature's process. And what is nature's process? First a germ, then an injury to the pharyngeal mucous membrane, then the epithelium being devitalized and partly destroyed, and contiguous blood vessels damaged; then an exudation upon the surface; then coagulation necrosis of tissue, the cells dying and becoming solidified or coalescing into a heeerogeneous mass containing within its meshes an admixture of walls of blood vessels, transfused lymph, pus corpuscles and epithelium called a false or pseudo membrane. Whether loosely adherent, or connected with the underlying structure by fibrous threads, the diseased and dead tissue in the process of repair is separated from the lining by a zone of cell infiltration.

And finally an inflammatory process is set up around the necrotic patch and the slough, if superficial, becomes infiltrated with pus and so loosened and cast off, the loss being made good by regeneration of epithelial cells beneath, or if large by cicatricial tissue. Sometimes sloughs become gangrenous; that is, micro-organisms penetrate the tissues and set up septic and putrid decomposition, and then it becomes grave, for decomposition and sepsis are added to the original disease. But even then suppurative inflammation may lead to separation of dead tissue and repair. Now, if the serum aids nature, its effects are either constitutional, antagonizing the effect of the toxines in the system, or they go farther and lift the incubus from the vaso-motor centers, stimulate diapedesis, and aid suppurative inflammation in the separation of the slough and the restoration of the mucous membrane to its integrity.

Schumaker in his latest edition enumerates some accidents following the injections, as "erythema, urticaria, fever, swollen glands, arthritis, hæmaturia and albuminuria. They have also been thought to favor an increased tendency to paralysis. Several deaths have been reported directly following this method of treatment. Its value cannot yet be estimated. Nevertheless the statistics collected are very suggestive."

Cold Air for Consumptives.

Modern Medicine and Bacteriological Review.

There are now six sanitaria in Germany at which consumptives are treated by the cold-air method first suggested by Detweiler. The principal idea of this method of treatment is that the patient should be exposed constantly to air at a low temperature, currents of cold air being allowed to pass through the bedroom at night and during the day as much of the time being spent in the open air as possible. The pure cold air quiets the cough, lessens the temperature, arrests night sweats, improves the appetite, and remedies or arrests the course of the disease. Observations made in these sanitaria have developed the interesting fact that it is exceedingly rare indeed that infection of healthy persons by contact with consumptive inmates is observed. The probable reason for this is that greater precautions are taken to prevent infection in the sanitaria where especial attention is given to this disease than in ordinary hospitals, or indeed in private houses. It is well known that the breath of a consumptive does not contain bacilli; It is only when sputum has been dried and reduced to powder so that it is carried about by the air that the possibility of infection occurs.

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Topeka, Kansas, Saturday, Nov. 30, 1895.

Education and Medicine.

The benefits of a good education in the study of medicine are now so generally appreciated that any extensive discussion of the subject seems useless. There seems to be a tendency among those high in the scale of medical education to accept rather the title of accomplishment itself. There are many young men and young women who can boast a degree of B.S. or A.B., but who when put to the test are poorly equipped for the study of medicine. A mind trained to study will, of course, find the work much easier and perhaps reach a higher rank in the class room, but there is another element necessary to the construction of a complete medical student, the ability to make a practical application of the facts and theories taught. Would it not be better to make the standard of requirements correspond to the necessary knowledge of the various subjects without regard to the time of study or the degrees.

Literary degrees do not always indicate something earned, and it is an unfortunate fact that the higher degrees are usually honorary.

It is the custom of many colleges to confer the degree of A.M. upon graduates who have attained a certain amount of eminence in literary work. Very frequently the degree is conferred upon people of note who have had no college work at all, and so with most of the higher college degrees. The degrees of A.B. and B.S. are always earned and indicate that a certain amount of work has been done. It is always true, however, that the man who sports an A.M. or Ph.D. after his name takes a higher place in the world of authors than the A.B. or B.S.

Certain degrees that are conferred upon men of learning have a certain commercial value and the degree of M.D. has a commercial value dependent upon the surroundings and ability of the individual holding it. The value seldom depends upon the institution granting the degree in spite of the fact that it costs more in both time and money in some institutions than others. No matter how little of medicine a man may really know, if he possesses a degree of M. D. he is accorded all the respect and all the credit to which the degree entitles him, and from a commercial standpoint it is worth as much to him coming from the most insignificant college in the country as if it were granted by a great university.

At some future time it is possible that there will be great universities of medicine where each department of medicine may be taught and where different degrees may be awarded for proficiency in different lines. The commercial value of the degree of M.D. will then be somewhat modified by the number of additional titles one has earned. It is a total impossibility for a single mind to take in in its entirety the whole subject of medicine as it is known today, and the degree of M.D. denotes nothing more at this time than that the possessor is ready to enter upon the study of this vast science.

The Antidote for Carbolic Acid.

New York Medical Times.

Probably there are more accidental deaths from carbolic acid than from all other poisonous drugs combined. The writer makes this estimate from newspaper reading, never having attempted any compilation of exact statistics. At any rate, the tragic story has been of late repeated with startling frequency. When the number of deliberate suicides from carbolic acid has been added to the foregoing, the total is so large that it demands attention.

What causes this state of affairs, and how shall it be remedied?

First, the drug is handy. Chloroform, prussic acid and morphine cannot be obtained without a physician's prescription; they are not commonly to be found in the house; everybody knows them to be highly dangerous. Not so with carbolic acid. It is sold freely to all who ask for it, labeled "poison" to be sure, but without further restriction. The people are familiar with it in its simple form and in its combinations. They have been taught to consider it a valuable antiseptic of wide applicability. They apply it, in dilution, to various lesions of the body, and flush the waste pipes of the house with the crude material. It stands on the shelf. They are not particularly afraid of it. No wonder that the ignorant and careless suffer accidentally from it with great frequency, and that the angry and malicious avail themselves of the opportunities for evil it affords.

Obviously it should be as difficult to obtain carbolic acid as prussic acid. In lethal quantities, the former shows results almost as soon as the latter. The anæsthetic property of carbolic acid adds to its danger. Then let it not be commonly used for antisepsis. Stop familiarizing people with it. Teach them that it is too dangerous to have around.

What shall be done in case of accident from it? For a long time the profession stood helpless. Even now a majority of physicians are ignorant of the antidote. On two occasions the writer has brought this to the notice of his professional brethren, and thus spread knowledge which should be taught in the colleges and become common property. Surely this Society, with its widely circulating reports, will accomplish the desired result.

The antidote to carbolic acid is simple and to be had in every well ordered household. Knowledge of its specific worth came by accident. One day while making some experiments with the pure acid, an unlucky movement sent two ounces of it upon my hand. In about two seconds I had it under a stream of water and washed it well, but to no purpose; it became white and numb. There seemed to be no escape from the usual result—desquamation and slow recovery of the sense of touch. But the odor was persistent and unpleasant. In the belief that it might be changed thereby, a servant was sent to the kitchen for a cup of cider vinegar. While bathing and rubbing the affected parts with vinegar, what was my amazement to behold a complete restoration of color and function! In five minutes nothing remained in evidence except the modified odor.

That was the beginning. Numerous clinical verifications were obtained later, but considerable time elapsed before evidence was obtained as to the antidotal action of vinegar when the mucous membrane was affected. It came from our colleague, C. S. Kinney, M.D., and this is his communication:

HOSPITAL FOR THE INSANE,
MIDDLETOWN, N. Y., December, 1893.

"At 7 o'clock in the morning of August 4, 1884, a nurse called me to see a man who had swallowed some carbolic acid. The patient was found with his lips, mouth and tongue coated white where the acid had touched them, and the strong characteristic odor of the acid was present. He was at once given a half cup of vinegar diluted with an eqnal amount of water, and this followed in a few moments by a second dose of vinegar and water. As the time hung heavily on my hands while waiting for the stomach pump, the patient was given some

milk, which he willingly drank. The odor and the discoloration from the acid had disappeared from the patient's lips, mouth and tongue on taking the vinegar and water and on using the stomach pump no odor from the liquid that pumped from his stomach could be detected. After the stomach had been carefully washed out the patient was fed with hot milk for several days, and no further symptoms developed.

"It was not until May, 1887, that I noticed in the *Recorder* an article which had been presented before the International Hahnemannian Association, by Dr. Edmund Carleton, of New York, on the use of vinegar as an antidote. I have always thought I was indebted to him for the knowledge of the action of vinegar, as my acquaintance with Dr. Carleton antedated my use of vinegar as an antidote of carbolic acid by a number of years, and I may have heard it from him. Since seeing his explanation for the use of vinegar as an antidote for the acid, I have had an opportunity to test its efficiency in a number of instances, and have always found it to be reliable in every particular; and in no instance where the vinegar has been used within a few moments has there been any eschar formed."

That completes the chain. Cider vinegar is the antidote to carbolic acid. It is a fair inference that acetic acid of the shops will produce a similar action. Experiments will show.

Not wishing to divert attention from the subject presented, I nevertheless would like, in addition, to mention a hint received from observation of the pure effects of carbolic acid. It is a hint in the direction of materia medica and therapeutics, and is this: The bleaching and anæsthesia are somewhat similar to those of leprosy.

Pilocarpine as a Prophylactic in Diphtheria.

The Sanitarian.

In a late number of the *Wiener Medicinische Presse*, Dr. Sziklai reports to the Congress of Physicians and Naturalists of Hungary and to the Congress of Rome on the action of pilocarpine in diphtheria. He has shown that this remedy is not only a certain and rapid cure for the disease, but that it may be used as a pophylactic in epidemics of diphtheria, especially in homes in which a number of the family is already attacked with the disease. The prophylactic properties of pilocarpine are especially remarkable in croup and in pseudo-diphtheritic affections of the larynx.

Kovacs, Vas, many others have employed pilocarpine with success in pneumonia and in croup.

Szilklai uses pilocarpine in cases in which diphtheria is present, but he recommends its use as a preventive, for:

1. There is never any certainty that an epidemic of diphtheria is not threatened, and that the second case in the same family may or may not be polymicrobic.

2. Pilocarpine may, even in true diphtheria, prevent the formation of obstructive false membranes, a fact of which the author has on many occasions had an opportunity to be convinced. He also recommends pilocarpine as a prophylactic to be prescribed in doses as follows:

One per cent solution of hydrochlorate of pilocarpine to be administered three times a day, in ten-drop doses. For children under one year, half of that dose will be sufficient.

The author has never had any accident from the use of pilocarpine. It is true, he has always used the greatest caution in the use of the medicine. During the last three and a half years Sziklai has used this remedy constantly, either as a curative agent or a prophylactic, and sometimes, too, in large doses, exceeding largely that laid down in the *Codex*, without having met any symptom that caused him the least anxiety; 16 drops of distilled water represents about 1 gram. The daily prophylactic dose of a solution of no injurious effect on a child over one year of age.

The same dose has been used by Professor Stiller, of Budapest, for another purpose, and also the least danger. It is by relying on an author so competent as the distin-

guished professor that the author has prescribed the remedy in such large doses.

To prevent the outbreak of an epidemic, prophylactic remedies must be used, especially those that have the reputation of specifics againt given diseases, which they are known to cure certainly and speedily.

Pilocarpine is a specific not only against diphtheria, but also against pseudo-diphtheria; it cures these diseases with certainty and rapidity; it is a specific against malaria. As quinine is useful as a prophylactic against intermittent fever, pilocarpine is equally useful against diphtheria alone or against polymicrobia or against pseudo-diphtheria.

The author cites many cases in support of his thesis, and shows that, even in the absence of hygienic care, the children in which a number has been already attacked during the course of an epidemic may be preserved from infection.

The Topeka Academy of Medicine and Surgery.

The Topeka Academy of Medicine and Surgery met in A. O. U. W. hall Nov. 11, 1895, with Dr. Magee in the chair.

The members present were Drs. Mitchell, Lewis, W. E. McVey, R. E. McVey, Ward, Powell, Beers, Magee, Barnes, Adams and Sheldon.

The minutes were read and approved.

Dr. John E. McCuaig was elected a member of the society.

The application of Dr. C. C. Seabrook was referred to Drs. Ward, W. E. McVey and Lewis, who deferred their decision until a month later in order to ascertain whether Dr. Seabrook was a resident of Shawnee county.

Dr. Magee delivered the president's annual address, which was followed by election of officers for the ensuing year.

Dr. M. B. Ward was elected president, Dr. W. E. McVey, vice-president and Dr. Ida C. Barnes, secretary and treasurer.

It was moved and carried that secretary notify members in arrears according to the constitution.

At the next meeting papers will be read by Dr. Whitmer on "Massage;" Dr. Longshore, on "Tonsilitis," and Dr. Ward will give reports of some surgical cases.

Adjourned to meet second Monday in December. IDA C. BARNES, M. D., Sec.

On the Relationship Between Auto-Intoxication and Neurasthenia.

North Carolina Medical Journal.

Dr. Alvis Pick (*Wiener klinische Rundschau*,) quotes Baumann's discovery of the presence of certain aromatic bodies (indol, scatol, paracresol, phenol, etc.,) in the urine of man and some animals, and discusses at length their mode of production in the body. Indol, which he takes as a type, is found in such conditions as intestinal obstruction, atony, paralysis, etc. It is eliminated partly in the fæces and partly in the urine. These bodies act as poisons in the blood and produce auto-intoxication. The manner in which auto-intoxication is modified or prevented is next discussed, stress being laid on the anti-putrefactive properties of the HCl in the gastric juice. The biliary and pancreatic juices also have a preventive property, so also have the fatty acids, resulting from splitting up of the carbohydrates. If these national safeguards fail, blood-poisoning results. Uræmia is possibly a form of autoxication. Possibly this may resnlt from an excess of normal urinary constituents, but Van Noordeen states that uræmic poisons do not exist in the normal constituents, "but that, primarily from reaction upon the tissue in retention, opportunity is given for the production of poisonous substances from natural metabolic products."

The author next discusses the *modus operandi* of certain articles of food (shell-fish, strawberries, etc.) on some persons as poisons. There is reflex action, but this is the path of morbid action, not its cause. There must be (1) individual predisposition; (2) poison introduced into the blood (*a*) improper ingesta (decomposing

sausage, fish, etc) or (*b*) it may originate in the intestine from the combined action of putrefaction and certain micro-organisms. Similar symptoms may arise from such drugs as antipyrin, iodoform, etc. In these cases the skin is often affected (erythema, urticaria, etc.), but other organs are also affected. Hence, some cases of pernicious anæmia, originating in gastrectasy, may be cured by lavage of the stomach.

Auto-intoxication produces marked nervous phenomena, which may be classified thus:—1. Giddiness; sometimes a true vertigo *a stomacho læso*. 2. Headache; this is common in habitual constipation with urine loaded with indican. 3. Heaviness and sense of pressure on head. 4. Cerebral vomiting due to poisoning of the medulla. 5. Convulsions. The author had a patient who had convulsions soon after meals, and was cured by antifermentative treatment. 6. Psychical disturbances. Delirium with normal pulse and temperature. Insanity is often associated with obstipation, and is relieved when this is cured. Irritable temper and hypochondriasis are other symptoms. 7. Peripheral nervous symptoms—such as tremor. There may be all the symptoms of Basedow's disease, and the author believes that this affection may sometimes originate in auto-intoxication. These symptoms, which may arise singly, in association, or one after the other, afford a picture of true neurasthenia, and Pick believes that many, though not all, cases of neurasthenia originate in auto-intoxication.

The author refers to the most frequently alleged causes of auto-intoxication—*e. g.* over-fatigue, sexual excesses, mental anxiety, etc.; and adds that the disease is more common among indigent than among energetic persons.

He next discusses the pathogeny of auto-intoxication, and says that in medicine it plays a similar *role* to wound-infection in surgery.

In the third article treatment is discussed. Diet is of most importance. Food rich in albumen and carbohydrates should be given, and milk is very useful. Generally a mixed diet of eggs, flesh, milk, vegetables and farinaceous food is to be recommended. Therapeutics are directed by etiological considerations. Sluggish gastric movements should be stimulated by massage, electrical abdominal sponging, douching, etc. Internal antisepsis should be aimed at, and not calomel (gr. ss to die) is useful in this way. The intestinal tract must be kept clear by purgatives, lavage, massage, etc. Antifermentatives, such as creosote, menthol, guaiacol, etc., are advisable.

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